Exhibit 6

Specific Causation Expert Report for Edward Raymond

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Additionally, I reviewed the specific causation reports of Dr. Hatten and Dr. Bird regarding Mr. Raymond's level of exposure, which found that Mr. Raymond was exposed to levels that have been shown to be hazardous to human health, specifically bladder cancer.

Altogether, Dr. Hatten, Dr. Bird and Dr. Reynolds established Mr. Raymond had a substantial exposure to TCE and Benzene, and the level of his exposure exceeded the levels that have been shown to be hazardous to human health – specifically bladder cancer.

Bladder Cancer Associated with TCE and Benzene Exposure

I have read the general causation reports of Dr. Bird, Dr. Culp, Dr. Gilbert, Dr. Hatten and Dr. Plunkett. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of TCE, and Benzene and bladder cancer. These reports are consistent with my review of the literature and support my opinions on this case.

My review of the literature supports that exposure to TCE and Benzene are a risk factor for development of bladder cancer.

Trichloroethylene (TCE)

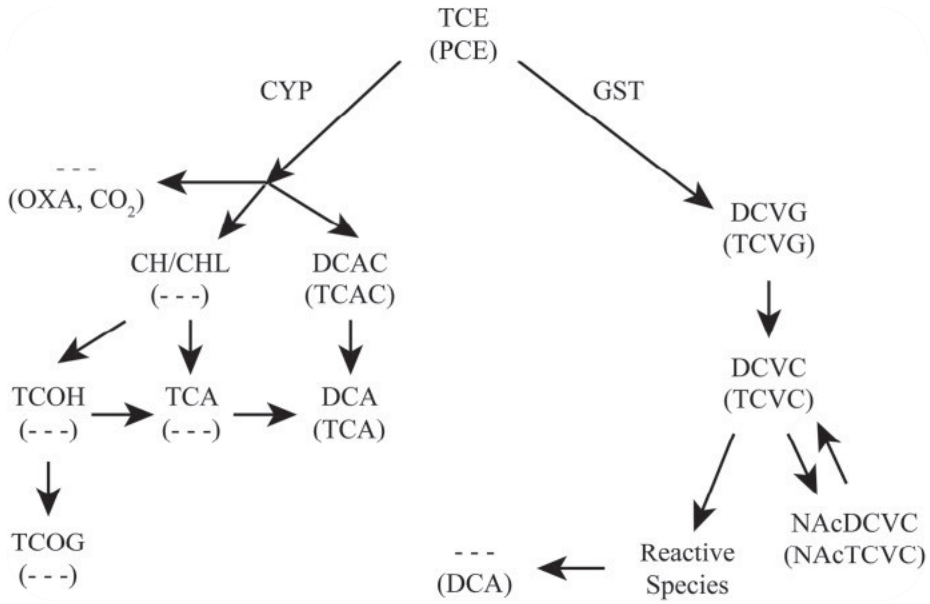
TCE was a solvent found in high concentrations at MCB Camp Lejeune. TCE has been designated as a carcinogen by the IARC and the evidence linking TCE to bladder cancer is suggestive. The following Cohort studies, Raaschou-Nielson et al, (2003), and Hansen et al (2013), showed increased risk of bladder cancer in workers exposed to TCE. The Cancer Hazard of TCE was evaluated by IARC (2014) the US EPA (2001b) National Toxicology Program, (2015). The conclusion of all three assessments was that there is sufficient evidence that TCE is a human carcinogen.

The carcinogenic pathway of TCE is similar to PCE. TCE is metabolized by oxidation into either cytochrome 450 or via glutathione conjugation into genotoxic metabolites. TCE exposure is primarily through inhalation and secondary through dermal. This is because TCE is not water soluble (Cichoki et al, 2016).

TCE is highly fat soluble and is readily taken up into fat tissue. TCE and PCE have similar metabolic pathways (see Fig 7). They are either broken down into toxic metabolites by oxidation in the liver or conjugation in the kidney.

In the oxidation of TCE, the chemical is metabolized through several steps by the Cytochrome P 450 enzyme, CYP2E1 to multiple substrates that eventually end in Dichloroacetic Acid (DCA), Trichloroethanol (TCOH), and Trichloroacetic Acid (TCA). In the Conjugative pathway TCE is then conjugated with Glutathione S-Transferase that is metabolized to di and trichlorovinyl-L-cysteine. (DCVC and TCVC) and these can further metabolize to Mercapturic Acid.

Fig 7. (Cichoki 2016)



TCE is absorbed via the lungs, dermally and to a lesser degree orally. It is subsequently absorbed in the blood stream or stored fat tissue that causes a slower and delayed release of the compound that is metabolized via oxidation through the liver in the oxidation pathway and to a lesser degree conjugation with GST into toxic metabolites, DCA, TCA and or TCOH. It is also taken up into the kidneys where it is conjugated into toxic metabolites and DCA and Mercapturic acid where it can subsequently be excreted in the urine.

Genotoxin is a chemical or agent that causes DNA or chromosomal damage. TCE has been shown to be genotoxic. (Tabrez, 2009) Damage in germ cells causes germline or heritable altered traits and DNA damage in a somatic cell may result in somatic mutation that can lead to malignant transformation. (Tung et al. 2012) Metabolism of TCE via the oxidation pathway and conjugation pathway produces toxic metabolites that are genotoxic. Studies have shown that TCE has a strong association with kidney cancer. As TCE is metabolized through the kidney and produces toxic metabolites it creates chronic irritation, and genotoxic effects to renal tubules and subsequently increase risk of Renal cell Carcinoma. (Cichoki et al. 2016) These metabolites are further secreted in the urine and presented to the bladder. Furthermore, with chronic exposure daily, and TCE being lipophilic both of which gives chronic exposure to the urinary tract irrespective of water samples and these together allow this genotoxic drug to be exposed to the bladder urothelial.

Figure 8 (Lash et al. 2000)

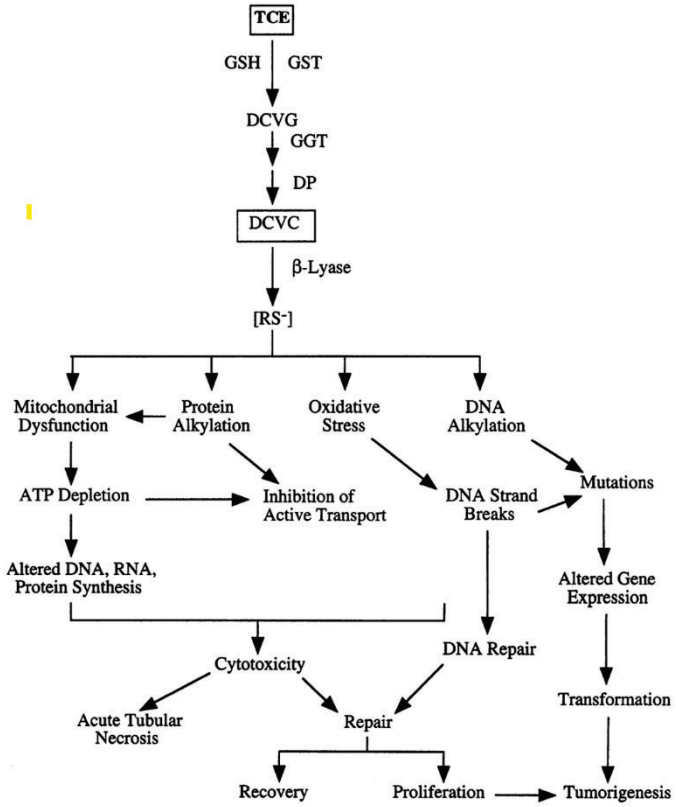


Figure 4. Summary scheme of the postulated modes of action of TCE via the GSH conjugation pathway for nephrotoxicity and nephrocarcinogenicity. The scheme summarized demonstrated and hypothesized modes of action of TCE in mammalian kidney, showing the various intracellular targets and the interplay between them in ultimately causing nephrotoxicity or nephrocarcinogenicity. Abbreviations used: DP, dipeptidase; RS⁻, reactive thiol and subsequent species generated from β-lyase-catalyzed metabolism of DCVC.

Urine that leaves the kidney travels through the ureters attached to each kidney and enters the bladder and stored until it is voided. The transient time can be hours. It is believed that the toxic metabolites that create the renal toxicity, genotoxicity and mutagenicity in them can have the same effect on the bladder urothelial, especially with longer storage times.

As discussed above, EPA finalized a rule banning TCE under the Toxic Substances Control Act, describing TCE as "extremely toxic." Dr. Bird provided a supplemental report that highlights the EPA's decision "that any lesser restrictions on the use of TCE would fail to adequately protect public health." (89 Fed. Reg. at 102572)

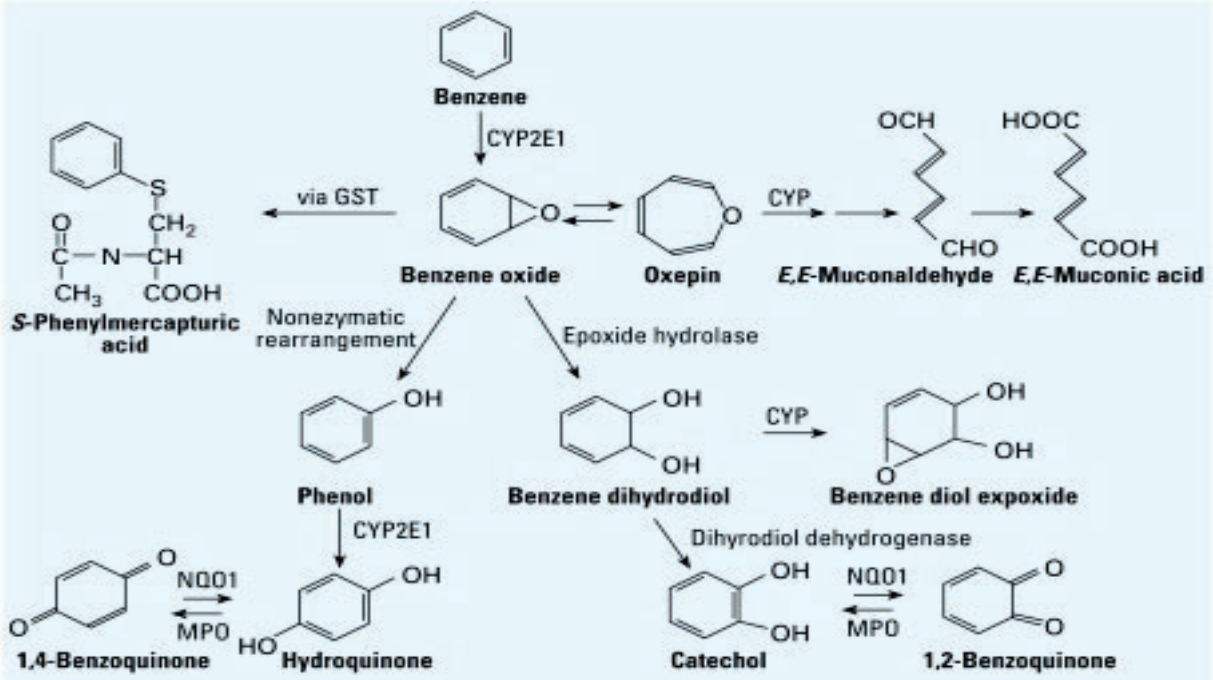
Based upon my education, training and experience, and considering the data and information identified and reviewed, it is my opinion to a reasonable degree of medical certainty that it is at least as likely as not that exposure to TCE from contaminated water at Camp Lejeune was hazardous to human health generally, that exposure to the Camp Lejeune water contaminated with TCE specifically is hazardous to human health, and, further, that the human health hazard could include the development of bladder cancer.

Benzene

Benzene has been classified as "carcinogenic to humans" by IARC (2012, 2018), as "carcinogenic in humans by all routes of exposure" by EPA (1998), and as "a known human carcinogen" by NTP (2021c). A recent epidemiologic study by Shala et al (2023) on male offshore petroleum workers showed that there was an increased risk of bladder cancer with benzene exposure with a HR 1.89 and cumulative benzene exposure HR of 1.6.

Benzene metabolism and its metabolites have genotoxic effect on bladder tissue that subsequently forms bladder cancer. Benzene is oxidized by the cytochrome P450 enzyme to benzene oxide. Benzene oxide is either oxidized by the Cytochrome P450 pathway to phenol which can be excreted or further oxidized by the Cytochrome P450 pathway (CYP2E1) to further metabolites to 1,4Benzoquinone or Hydroquinone. Furthermore, Benzene oxide can be oxidized via Cytochrome P450 liver enzymes to Catechol and its metabolites. which represents 70-85% and other metabolites represent the remainder.

Fig. 9 (Rappaport et al, 2009)



Genotoxin is a chemical or agent that causes DNA or chromosomal damage. (Phillips 2009) Damage in germ cells causes germline or heritable altered traits and DNA damage in a somatic cell may result in somatic mutation that can lead to malignant transformation. (Tung et al. 2012) Exposure to Benzene metabolites in particular Benzoquinone has shown in animal studies have been associated with numerous forms of genotoxic damage, including chromosome aberrations, sister chromatid exchanges, DNA and protein cross links, and DNA single and double strand breaks. These chromosome aberrations are associated with multiple forms of cancer. (Tung et al. 2012).

Mr. Raymond was exposed to this carcinogenic compound throughout his time at Camp Lejeune. He was exposed to a constant and persistent amount that tended to accumulate over time. The mechanism is that he had exposure through all three routes to include inhalation, oral and dermal. Once this chemical was absorbed it was then metabolized into metabolites either through the liver and oxidized cytochrome P-450 pathway or conjugated via conjugation by glutathione S transferase further creates toxic metabolites such as phenol and Benzoquinones.

Based upon my education, training and experience, and considering the data and information identified and reviewed, it is my opinion to a reasonable degree of medical certainty that it is at least as likely as not that exposure to benzene from contaminated water at Camp Lejeune was hazardous to human health generally, that exposure to the Camp Lejeune water contaminated with benzene specially is hazardous to human health, and, further, that the human health hazard could include the development of bladder cancer.

Specific Causation: TCE and benzene exposure and Edward Raymond's Bladder Cancer

As a Urologic Oncologist, I utilize differential etiology to provide the best patient care. In a differential etiology, a physician reviews known causes of a disease and attempts to rule out those causes as the cause of a patient's disease.

Bladder cancer can develop in patients with multiple risk factors. Environmental and occupational exposures are known risk factors for the development of bladder cancer. Exposures to multiple genotoxins, like smoking and workplace exposures, can have additive effects, acting together to contribute to the development of bladder cancer. Below is a discussion of known risk factors relevant to a differential etiology specific to Mr. Raymond's history.

Relevant Risk Factors for Developing Bladder Cancer

1. Exposure to Chlorinated Solvents and Carcinogenic Chemicals

As discussed throughout this report, there is overwhelming evidence that the chemicals present in the water at Camp Lejeune to which Mr. Raymond was exposed can cause bladder cancer. However, it is important to consider other potential exposures he may have had to possible carcinogenic chemicals.

Beyond the chlorinated solvents like those found at Camp Lejeune, some chemicals used in textiles, paint, and rubber manufacturing industries have been identified as associated with bladder cancer textile industry, rubber manufacturing industry, painting industry and aluminum and refined products industry have been shown to have an association with bladder cancer (IARC Monographs No. 100F, 2012)(Guha et al. 2010)(Singh and Chadha, 2016)

[27] Raymond Dep. 158:2-3
[28] Raymond Dep. 152:9-10